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     $0.60  Estimated cost this search
     $0.60  Estimated total session cost   0.166 DialUnits

File 410:The Chronolog 2009
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  File   5:Biosis Previews(R) 1926-2009/Sep W1
         (c) 2009 The Thomson Corporation
  File  73:EMBASE 1974-2009/Sep 09
         (c) 2009 Elsevier B.V.
*File  73: EMBASE Classic available to all Dialog customers.
See HELP NEWS 772 for information.
  File 155:MEDLINE(R) 1950-2009/Sep 11
         (c) format only 2009 Dialog
  File 399:CA SEARCH(R) 1967-2009/UD=15111
         (c) 2009 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

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E3        0 *AU=FRENTSCH MARCO ?
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E5        1  AU=FRENTSEL, I.
E6        1  AU=FRENTSEL, KH.
E7        1  AU=FRENTSEL' G-Y
E8        3  AU=FRENTSEL' KH
E9        1  AU=FRENTSOS J A
E10       1  AU=FRENTSOS J.A.
E11       1  AU=FRENTTE S
E12       2  AU=FRENTZ B

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E12       1  AU=ROTHE N VINGE

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isolat?)(20n)(antigen(w)specific)(20n)(t(w)cell? or t(w)lymphocyt? or cd4? or cd8?)
Processing
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         3352506  ISOLAT?
         1621823  ANTIGEN
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        14264231  CELL?
          884805  T(W)CELL?
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          579678  T(W)LYMPHOCYT?
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? rd s4
      S5          2  RD S4  (unique items)
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 5/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0019901393   BIOSIS NO.: 200700561134
Identification and isolation of murine antigen-reactive T cells according
  to CD154 expression
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk;
  Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold
  Alexander (Reprint)
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat
  Grp, Charitepl 1, D-10117 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de
JOURNAL: European Journal of Immunology  37 (9): p2370-2377 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200737322
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 5/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2009 The Thomson Corporation. All rts. reserv.

17794216   BIOSIS NO.: 200400161557
Direct characterisation of adenovirus-specific T helper (Th)-cells in
  healthy adult donors.
AUTHOR: Siegert Stefanie (Reprint); Rescher Ulrike; Chmielewicz Barbara;
  Frentsch Marco (Reprint); Ellerbrok Heinz; Radbruch Andreas
  (Reprint); Scheffold Alexander (Reprint); Thiel Andreas (Reprint)
AUTHOR ADDRESS: Klinische Immunologie, Deutsches Rheuma-Forschungszentrum,
  Berlin, Germany**Germany
JOURNAL: Blood  102 (11): p53b November 16, 2003 2003
```

MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology  San Diego, CA, USA  December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

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? s (cd154 or cd40L or cd40 ligand or gp39) (20n)(block? or suppress? or inhibit?
or prevent? or antagoni?) (20n)(diagnos? or detect? or
isolat?)(20n)(antigen(w)specific)(20n)(t(w)cell? or t(w)lymphocyt? or cd4? or cd8?)
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            4209  CD154
            9464  CD40L
            8686  CD40 LIGAND
             922  GP39
         1763121  BLOCK?
         1196060  SUPPRESS?
         5634636  INHIBIT?
         3137220  PREVENT?
         1462371  ANTAGONI?
         7027276  DIAGNOS?
         4015149  DETECT?
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         4144673  SPECIFIC
         2389965  T
        14264231  CELL?
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          228709  CD8?
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 7/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0020918436   BIOSIS NO.: 200900258770
Disruption of T Cell Suppression in Chronic Lymphocytic Leukemia by CD200 Blockade
AUTHOR: Pallasch Christian P (Reprint); Ulbrich Susanne; Brinker Reinhild; Uger Robert A; Hallek Michael; Wendtner Clemens-Martin
AUTHOR ADDRESS: Univ Cologne, Ctr Integrated Oncol Cologne Bonn, Dept Internal Med 1, Cologne, Germany**Germany
JOURNAL: Blood  112 (11): p721-722 NOV 16 2008 2008
CONFERENCE/MEETING: 50th Annual Meeting of the American-

Society-of-Hematology  San Francisco, CA, USA  December 06 -09, 2008; 20081206
SPONSOR: Amer Soc Hematol
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

7/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0020638860 BIOSIS NO.: 200800685799
Impact of myelin-specific antigen presenting B cells on T cell activation in multiple sclerosis
AUTHOR: Harp Christopher T; Lovett-Racke Amy E; Racke Michael K; Frohman Elliot M; Monson Nancy L (Reprint)
AUTHOR ADDRESS: Univ Texas SW Med Ctr Dallas, Dept Neurol, Dallas, TX 75390 USA**USA
AUTHOR E-MAIL ADDRESS: Nancy.Monson@UTSouthwestern.edu
JOURNAL: Clinical Immunology (Orlando)  128 (3): p382-391 SEP 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.clim.2008.05.002
ISSN: 1521-6616
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0020089819 BIOSIS NO.: 200800136758
Intracellular CD154 expression reflects antigen-specific CD8(+) T cells but shows less sensitivity than intracellular cytokine and MHC tetramer staining
AUTHOR: Han Young Woo; Aleyas Abi G; George Junu A; Yoon Hyun A; Lee John Hwa; Kim Byung Sam; Eo Seong Kug (Reprint)
AUTHOR ADDRESS: Chonbuk Natl Univ, Coll Vet Med, Dept Microbiol, Jeonju 561756, South Korea**South Korea
AUTHOR E-MAIL ADDRESS: vetvirus@chonbuk.ac.kr
JOURNAL: Journal of Microbiology and Biotechnology  17 (12): p1955-1964 DEC 2007 2007
ISSN: 1017-7825_(print) 1738-8872_(electronic)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0019901393 BIOSIS NO.: 200700561134
Identification and isolation of murine antigen-reactive T cells according to CD154 expression
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk; Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold Alexander (Reprint)
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat

Grp, Charitepl 1, D-10117 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de
JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200737322
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


19070219 BIOSIS NO.: 200600415614
The generation of thymus-independent germinal centers depends on CD40 but not on CD154, the T cell-derived CD40-ligand
AUTHOR: Gaspal Fabrina M C; McConnell Fiona M; Kim Mi-Yeon; Gray David; Kosco-Vilbois Marie H; Raykundalia Chandra R; Botto Marina; Lane Peter J L (Reprint)
AUTHOR ADDRESS: Univ Birmingham, MRC, Ctr Immune Regulat, Biomed Res Inst, Vincent Dr, Birmingham B15 2TT, W Midlands, UK**UK
AUTHOR E-MAIL ADDRESS: p.j.l.lane@bham.ac.uk
JOURNAL: European Journal of Immunology 36 (7): p1665-1673 JUL 2006 2006
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


18318845 BIOSIS NO.: 200510013345
Nonreplicating recombinant vaccinia virus expressing CD40 ligand enhances APC capacity to stimulate specific CD4+ and CD8+ T cell responses
AUTHOR: Feder-Mengus Chantal; Schultz-Thater Elke; Oertli Daniel; Marti Walter R; Heberer Michael; Spagnoli Giulio C; Zajac Paul (Reprint)
AUTHOR ADDRESS: Univ Basel Hosp, Res Ctr, ICFS, Lab 404,Dept Surg,Oncol Grp, Hebelstr 20, CH-4031 Basel, Switzerland**Switzerland
AUTHOR E-MAIL ADDRESS: pzajac@uhbs.ch
JOURNAL: Human Gene Therapy 16 (3): p348-360 MAR 05 2005
ISSN: 1043-0342
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


17847019 BIOSIS NO.: 200400217074
Blockade of CD40 pathway enhances the induction of immune tolerance by immature dendritic cells genetically modified to express cytotoxic T lymphocyte antigen 4 immunoglobulin.
AUTHOR: Sun Wenji; Wang Quanxing; Zhang Lihuang; Liu Yushan; Zhang Min; Wang Chunmei; Wang Jianli; Cao Xuetao (Reprint)
AUTHOR ADDRESS: Institute of Immunology, Zhejiang University, Hangzhou, 310031, China**China

AUTHOR E-MAIL ADDRESS: caoxt@public3.sta.net.cn
JOURNAL: Transplantation (Hagerstown)  76 (9): p1351-1359 November 15, 2003
 2003
MEDIUM: print
ISSN: 0041-1337
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

 7/3/8     (Item 8 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)


17794216   BIOSIS NO.: 200400161557
Direct characterisation of adenovirus-specific T helper (Th)-cells in
  healthy adult donors.
AUTHOR: Siegert Stefanie (Reprint); Rescher Ulrike; Chmielewicz Barbara;
  Frentsch Marco (Reprint); Ellerbrok Heinz; Radbruch Andreas (Reprint);
  Scheffold Alexander (Reprint); Thiel Andreas (Reprint)
AUTHOR ADDRESS: Klinische Immunologie, Deutsches Rheuma-Forschungszentrum,
  Berlin, Germany**Germany
JOURNAL: Blood  102 (11): p53b November 16, 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of
Hematology  San Diego, CA, USA  December 06-09, 2003; 20031206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

 7/3/9     (Item 9 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)


16548252   BIOSIS NO.: 200200141763
Normal Th1 development following long-term therapeutic blockade of
  CD154-CD40 in experimental autoimmune encephalomyelitis
AUTHOR: Howard Laurence M; Ostrovidov Serge; Smith Cassandra E; Dal Canto
  Mauro C; Miller Stephen D (Reprint)
AUTHOR ADDRESS: Department of Microbiology-Immunology, Northwestern
  University Medical School, 303 East Chicago Avenue, Chicago, IL, 60611,
  USA**USA
JOURNAL: Journal of Clinical Investigation  109 (2): p233-241 January, 2002
 2002
MEDIUM: print
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

 7/3/10     (Item 10 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)


14284021   BIOSIS NO.: 199800078268
Blockade of CD40-CD40 ligand pathway induces tolerance in murine contact
  hypersensitivity

AUTHOR: Tang Aimin; Judge Thomas A; Turka Laurence A (Reprint)
AUTHOR ADDRESS: Univ. Pa., 901-B Stellar-Chance Build., 422 Curie Blvd., Philadelphia, PA 19104-6100, USA**USA
JOURNAL: European Journal of Immunology 27 (12): p3143-3150 Dec., 1997 1997
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
? t s7/7/all

7/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0020918436 BIOSIS NO.: 200900258770
Disruption of T Cell Suppression in Chronic Lymphocytic Leukemia by CD200 Blockade
AUTHOR: Pallasch Christian P (Reprint); Ulbrich Susanne; Brinker Reinhild; Uger Robert A; Hallek Michael; Wendtner Clemens-Martin
AUTHOR ADDRESS: Univ Cologne, Ctr Integrated Oncol Cologne Bonn, Dept Internal Med 1, Cologne, Germany**Germany
JOURNAL: Blood 112 (11): p721-722 NOV 16 2008 2008
CONFERENCE/MEETING: 50th Annual Meeting of the American-Society-of-Hematology San Francisco, CA, USA December 06 -09, 2008; 20081206
SPONSOR: Amer Soc Hematol
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Suppression of patients' T cells is a key event in CLL pathogenesis and was demonstrated to be mediated by direct cell-cell contact of malignant CLL cells with T-cells. CD200 plays a critical role in regulating the immune system and has been shown to be up-regulated on the surface of different rumors including CLL. In this study we addressed the effects of CD200 over-expression on CLL cells on autologous T cells in a mixed lymphocyte reaction system. We used native and CD40 ligand (CD40L)-stimulated CLL cells as antigen-presenting cells (APCs) to expand autologous T cells of 14 patients T-cell proliferation was analyzed over 3 weeks of in vitroculture. A functional anti-CD200 antibody (IB9) was added to reveal CD200-mediated immunosuppression in the autologous system. Expansion of patient ***T*** -cells was assessed by flow cytometry including intracellular staining of FOXP3. Specificity towards CLL-specific antigens was monitored applying fibromodulin derived peptides for detection of specific T-cells by ELISPOT analysis. T-cell proliferation over 3 weeks of in vitro culture was significantly enhanced compared to control cells when using CD40L -stimulated APCs and an anti-CD200 antibody (p=0.0004). CD200 blockade was further shown to stimulate antigen-specific T-cell responses towards the F2 and F4 peptides of the CLL-associated antigen fibromodulin (p=0.04). Finally, the number of ***CD4*** +/CD25high/FOXP3+ T cells (T-reg) was significantly decreased in CD200 treated mixed lymphocyte reaction (p=0.04). In summary, CD200 blockade may provide therapeutic benefits in CLL by (I) enhancing T -cell expansion, (11) augmenting an antigen-specific ***T*** cell response with (III) suppression of regulatory T cells. CD200 seems to be an important immunosuppressive molecule in CLL: by CD200 blockade immune suppression can be overcome by altering tolerance

to tumor antigens and deregulation of regulatory T cells. This combination of an immune induction paralleled by a disruption of immunosuppressive factors makes anti-CD200 mAb a powerful tool for future treatment of CLL, possibly in combination with other

7/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0020638860 BIOSIS NO.: 200800685799
Impact of myelin-specific antigen presenting B cells on T cell activation in multiple sclerosis
AUTHOR: Harp Christopher T; Lovett-Racke Amy E; Racke Michael K; Frohman Elliot M; Monson Nancy L (Reprint)
AUTHOR ADDRESS: Univ Texas SW Med Ctr Dallas, Dept Neurol, Dallas, TX 75390 USA**USA
AUTHOR E-MAIL ADDRESS: Nancy.Monson@UTSouthwestern.edu
JOURNAL: Clinical Immunology (Orlando) 128 (3): p382-391 SEP 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.clim.2008.05.002
ISSN: 1521-6616
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The rote of B cells in the pathogenesis of Multiple Sclerosis (MS) is incompletely understood. Here we define a possible role for B cells as myelin-specific antigen presenting cells (B-APCs) in MS. Peripheral blood B cells (PBBC) isolated from both MS patients and healthy controls (HC) were activated in vitro with either CD40L/IL-4 or a Class B CpG oligodeoxynucleotide (CpG CDN)/IL-2. Both activation techniques induced PBBCs to upregulate CD80 and HLA-DR, rendering them more efficient APCs than resting 8 cells. Although the ***CD40L*** /IL-4 B-APCs were highly effective in eliciting CNS-antigen specific proliferation by autologous T-cells, CpG ODN/IL-2 stimulated B cells were not. Furthermore, ***CD40L*** /IL-4 B-APC induced responses by autologous CD4(+) T cells were susceptible to blocking with anti-HLA-DR antibody, suggesting that T cell responses were specific for antigen presentation by B-APC. CNS-antigen specific CD8(+) T cell proliferation was also blocked by HLA-DR, suggesting that CD8(+) proliferation is in part dependent on ***CD4*** (+) help. (C) 2008 Elsevier Inc. All rights reserved.

7/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0020089819 BIOSIS NO.: 200800136758
Intracellular CD154 expression reflects antigen-specific CD8(+) T cells but shows less sensitivity than intracellular cytokine and MHC tetramer staining
AUTHOR: Han Young Woo; Aleyas Abi G; George Junu A; Yoon Hyun A; Lee John Hwa; Kim Byung Sam; Eo Seong Kug (Reprint)
AUTHOR ADDRESS: Chonbuk Natl Univ, Coll Vet Med, Dept Microbiol, Jeonju 561756, South Korea**South Korea
AUTHOR E-MAIL ADDRESS: vetvirus@chonbuk.ac.kr
JOURNAL: Journal of Microbiology and Biotechnology 17 (12): p1955-1964 DEC 2007 2007
ISSN: 1017-7825_(print) 1738-8872_(electronic)

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A recent report showed that analysis of CD 154 expression in the presence of the secretion inhibitor Brefeldin A (Bref A) could be used to assess the entire repertoire of antigen-specific CD4(+) T helper cells. However, the capacity of intracellular CD 154 expression to identify antigen-specific CD8(+) T cells has yet to be investigated. In this study, we compared the ability of intracellular CD154 expression to assess antigen-specific CD8(+) T cells with that of accepted standard assays, namely intracellular cytokine IFN-gamma staining (ICS) and MHC class I tetramer staining. The detection of intracellular CD154 molecules in the presence of Bref A reflected the kinetic trend of antigen-specific CD8(+) T cell number, but unfortunately showed less sensitivity than ICS and tetramer staining. However, ICS levels peaked and saturated 8 h after antigenic stimulation in the presence of Bref A and then declined, whereas intracellular CD154 expression peaked by 8 h and maintained the saturated level up to 24 h post-stimulation. Moreover, intracellular CD 154 expression in antigen-specific CD8(+) T cells developed in the absence of CD4(+) T cells changed little, whereas the number of IFN-gamma-producing CD8(+) T ***cells*** decreased abruptly. These results suggest that intracellular CD154 could aid the assessment of antigen-specific CD8(+) T cells, but does not have as much ability to identify heterogeneous ***CD4*** (+) T helper cells. Therefore, the combined analytical techniques of ICS and tetramer staining together with intracellular CD154 assays may be able to provide useful information on the accurate phenotype and functionality of antigen-specific CD8(+) T ***cells*** .

7/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


0019901393 BIOSIS NO.: 200700561134
Identification and isolation of murine antigen-reactive T cells according to CD154 expression
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk; Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold Alexander (Reprint)
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat Grp, Charitepl 1, D-10117 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de
JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1002/eji.200737322
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: T helper (Th) cells are central regulators of adaptive immune responses. However, the ***detection*** of the small number of Th cells specific for a particular antigen or pathogen is still a major challenge. CD154 was recently introduced as a marker for antigen-specific Th cells. To date, this technology was not applicable for mice - arguably the most important immunological model system. CD154 is difficult to detect due to its rapid removal from the cell surface upon binding to ***CD40*** during ***antigen*** - ***specific*** activation by APC. We present an efficient strategy to block the degradation of murine CD154 by

combined use of antibodies against ***CD40*** and CD154. This strategy makes CD154 easily accessible for surface staining, which allows isolation and expansion of rare ***antigen*** ***specific*** Tcells. Importantly, CD154 identified all specific Tcells in strongly Th1- or Th2-polarized immune responses against pathogens like Salmonella typhimurium and Heligmosomoides polygyrus, independent of their potential to produce cytokines. We demonstrate that ***CD154*** can in fact be used as a reliable marker for antigen-specific CD4 T cells in mice, offering a unique option to analyze, isolate and rapidly expand the entire pool of Th-cells generated during a physiological ***T*** ***cell*** response in vivo.

7/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


19070219 BIOSIS NO.: 200600415614
The generation of thymus-independent germinal centers depends on CD40 but not on CD154, the T cell-derived CD40-ligand
AUTHOR: Gaspal Fabrina M C; McConnell Fiona M; Kim Mi-Yeon; Gray David; Kosco-Vilbois Marie H; Raykundalia Chandra R; Botto Marina; Lane Peter J L (Reprint)
AUTHOR ADDRESS: Univ Birmingham, MRC, Ctr Immune Regulat, Biomed Res Inst, Vincent Dr, Birmingham B15 2TT, W Midlands, UK**UK
AUTHOR E-MAIL ADDRESS: p.j.l.lane@bham.ac.uk
JOURNAL: European Journal of Immunology 36 (7): p1665-1673 JUL 2006 2006
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In this report, we show that the formation of germinal center (GC)-like structures to thymus-independent type 2 antigens in mice depends on intact signals through CD40, but does not depend on T cell-derived CD40-ligand (CD 154). In addition, we show that follicular dendritic cells (FDC) are also critical to thymus-independent GC formation, as their depletion by blockade of lymphotoxin-P receptor signals abrogated GC development unless the responding B cells bound antigen with high affinity. Further evidence that immune complexes drove this CD40-dependent B cell proliferation was provided by the observation that an antibody that detects immune complexes containing complement component 4 on FDC also inhibited thymus-independent GC formation when injected in vivo at the time of immunization. Finally, we show that thymus-independent B cell proliferation was associated with class switching to IgG3, as IgG3(+) antigen-specific switched B cells could be visualized directly in GC, suggesting that immune complexes can provide the signals for class switching within GC in the absence of ***CD154*** .

7/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


18318845 BIOSIS NO.: 200510013345
Nonreplicating recombinant vaccinia virus expressing CD40 ligand enhances APC capacity to stimulate specific CD4+ and CD8+ T cell responses
AUTHOR: Feder-Mengus Chantal; Schultz-Thater Elke; Oertli Daniel; Marti Walter R; Heberer Michael; Spagnoli Giulio C; Zajac Paul (Reprint)
AUTHOR ADDRESS: Univ Basel Hosp, Res Ctr, ICFS, Lab 404,Dept Surg,Oncol

Grp, Hebelstr 20, CH-4031 Basel, Switzerland**Switzerland
AUTHOR E-MAIL ADDRESS: pzajac@uhbs.ch
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ABSTRACT: Recombinant poxviruses expressing immunomodulatory molecules together with specific antigens represent powerful vaccines for cancer immunotherapy. Recently, we and others have demonstrated, in vitro and in vivo, that coexpression of CD80 and CD86 costimulatory molecules enhances the immunogenic capacity of a recombinant vaccinia virus (rVV) encoding different tumor-associated antigens. To further investigate the capacity of these vectors to provide ligands for different costimulatory pathways relevant in the generation of T cell responses, we constructed a recombinant virus (rVV) expressing CD40 ligand or CD154 (CD154rVV). Upon binding the CD40 receptor expressed on antigen presenting cells (APC), this molecule, physiologically expressed on activated CD4(+) T cells, increases their antigen presentation and immunostimulatory capacities.Therefore, we evaluated the effects of CD154rVV infection on APC activation and its consequences on T cell stimulation. CD154rVV infection of autologous fibroblasts, monocytes, or iDC promoted the expression of a number of cytokines, including GM-CSF, TNF-alpha, and IL-15 in iDC. Most importantly, IL-12 p40 gene expression and protein secretion were induced by CD154rVV but not by wild-type VV (WT VV) in either CD14(+) cells or iDC, and these effects could be blocked by anti-CD40 monoclonal antibodies. Furthermore, phenotypic characterization of CD154rVV infected iDC revealed enhanced expression of CD83 and CD86 surface markers as compared with wild-type vaccinia virus infection. As expected, VV infection triggered cytokines gene expression in cultures including APC and T cells from VV immune donors. However, cytokine genes typically expressed by T cell receptor triggered T cells such as those encoding IL-2 and IFN-gamma, or T cell proliferation, were detectable to a significantly higher extent in CD154rVV infected cultures, as compared with WT VV.Activation of specific ***CD8*** (+) T cells was then investigated using MART-1/Melan-A(27-35) epitope as the model of tumor-associated antigen (TAA). In the presence of CD154rVV activated APCs, significantly higher numbers of specific cytotoxic CD8(+) T cells were detected, as compared with cultures performed in the presence of WT VV or in the absence of virus.Taken together, these data indicate that functional CD154 expression from rVV infected cells promotes APC activation, thereby enhancing antigen-specific T cell generation. Such a recombinant vector might help bypass the requirement for activated helper cells during CTL priming, thus qualifying as a potentially relevant vector in the generation of CD8(+) T ***cell*** responses in cancer immunotherapy.

7/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


17847019 BIOSIS NO.: 200400217074
Blockade of CD40 pathway enhances the induction of immune tolerance by immature dendritic cells genetically modified to express cytotoxic T lymphocyte antigen 4 immunoglobulin.
AUTHOR: Sun Wenji; Wang Quanxing; Zhang Lihuang; Liu Yushan; Zhang Min; Wang Chunmei; Wang Jianli; Cao Xuetao (Reprint)
AUTHOR ADDRESS: Institute of Immunology, Zhejiang University, Hangzhou,

310031, China**China
AUTHOR E-MAIL ADDRESS: caoxt@public3.sta.net.cn
JOURNAL: Transplantation (Hagerstown) 76 (9): p1351-1359 November 15, 2003 2003
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LANGUAGE: English

ABSTRACT: Background. Immature dendritic cells (DCs) have the tolerogenic potential to induce alloantigen-specific immune tolerance. Cytotoxic T lymphocyte antigen 4 immunoglobulin (CTLA4Ig) gene-modified immature DCs have been shown to maintain their tolerogenicity and prolong allograft survival to some extent. We investigated whether blockade of CD40 pathway by anti-CD40 ligand (L) monoclonal antibody (mAb) could enhance the immune tolerance induction by immature DCs genetically modified to express CTLA4Ig (DC-CTLA4Ig). Methods. The tolerogenic properties of DC-CTLA4Ig were analyzed. In the vascularized heferotopic heart transplantation murine model, 2X106 DC-CTLA4Ig were infused intravenously into recipients, with or without a concomitant administration of anti-CD40L mAb 7 days before transplantation. Host responses to donor alloantigen were quantified by mixed leukocyte reaction and CTL assays. Donor major histocompatibility complex class II (Iab) expression in recipient lymph nodes was detected posttransplantation by semiquantitative reverse transcriptase-polymerase chain reaction. Results. The allostimulatory activity of DC-CTLA4Ig was reduced. DC-CTLA4Ig also induced alloantigen-specific T-cell hyporesponsiveness and polarized T helper 2 cytokine production. Pretreatment of the recipients with DC-CTLA4Ig modestly prolonged allograft survival, without long-term allograft acceptance. Combined administration of DC-CTLA4Ig and anti-CD40L mAb significantly prolonged cardiac allograft survival, with long-term (> 100 days) survival of 50% of the allografts in the pretreated recipients. More potent donor-specific ***inhibition*** of immune response against alloantigens and increased microchimerism were observed in these recipients. Conclusions. ***Blockade*** of ***CD40*** pathway with anti-CD40L mAb potentiates the tolerogenic potential of DC-CTLA4Ig and enhances the induction of antigen-specific immune tolerance more effectively.

7/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


17794216 BIOSIS NO.: 200400161557
Direct characterisation of adenovirus-specific T helper (Th)-cells in healthy adult donors.
AUTHOR: Siegert Stefanie (Reprint); Rescher Ulrike; Chmielewicz Barbara; Frentsch Marco (Reprint); Ellerbrok Heinz; Radbruch Andreas (Reprint); Scheffold Alexander (Reprint); Thiel Andreas (Reprint)
AUTHOR ADDRESS: Klinische Immunologie, Deutsches Rheuma-Forschungszentrum, Berlin, Germany**Germany
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ISSN: 0006-4971
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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Background and Aims: Adenovirus (Ad) infections have been increasingly recognised as an important cause of morbidity and mortality in allogeneic stem cell transplant recipients, especially in children. Immune responses to Ad infection are not fully understood, but T-cell mediated immunity appears to be important for recovery. The target proteins of Ad and their epitopes are still unknown. The aim of the present study was to evaluate and characterise Ad-specific T-cell responses in healthy adult donors. Methods: T cell responses to different Ad serotypes were investigated in healthy adult donors using a short-term stimulation assay. Whole blood was stimulated for 6 hours with anti-CD28 and different antigens (e.g. Ad2, 3, 4, 7, 12 lysates, control lysates, SEB, CMV lysate) in the presence of the secretion inhibitor Brefeldin A. After stimulation, cells were stained for CD69, CD4 and TNF-alpha for FACS analysis. In order to assess the complete fraction of Ad-specific CD4+ Th-cells we also tested the expression of antigen-reactive CD154 (CD40L), as a marker for antigen -specific Th-cells while antigen-reactive TNF-alpha was used to evaluate proinflammatory Th-cells. Results: Ad-specific T cells reactive to at least one of the used lysates could be detected in all of the adult donors analysed. In response to Ad3 lysate 0.06% of CD4+ T-cells became CD69+/TNF-alpha+ (median; range 0.03-0.29%; n=13) compared with 0.01% for anti-CD28 alone or control lysate. Analysis of Ad3-reactive CD154+ expression always revealed slightly higher percentages as compared to TNF-alpha+ expression (0.1%; 0.04-0.79% vs. 0.06%; 0.03-0.29%, n=13). This indicates the feasibility of antigen-reactive CD154 expression after short-term in vitro stimulation for the assessment of the entire repertoire of Th-cells specific for a particular antigen or mixtures of antigens. Of interest, the frequency of specific T cells varied from donor to donor. For example, Ad7 lysate induced strong ***CD4*** + Th-cell responses only in selected donors. In one particular healthy donor nearly 5 in 1,000 CD4+ Th-cells (CD4+/CD69+/TNF-alpha+: 0.45% vs. 0.05% control lysate) reacted upon stimulation with Ad7. Conclusions: Our results confirm the previous assumption that Ad-specific Th-cells in human adults may be characterised by widespread crossreactivities for different Ad serotypes. However, since distinct Ad serotypes elicit immune responses only in selected adult donors further analysis of the fine specificities of these Th-cell responses are necessary. Moreover, this will be a prerequisite in order to define protective Ad-specific T-cell responses to develop protocols for specific adoptive immunotherapies in allogeneic stem cell transplantation.

7/7/9 (Item 9 from file: 5)


16548252 BIOSIS NO.: 200200141763
Normal Th1 development following long-term therapeutic blockade of CD154-CD40 in experimental autoimmune encephalomyelitis
AUTHOR: Howard Laurence M; Ostrovidov Serge; Smith Cassandra E; Dal Canto Mauro C; Miller Stephen D (Reprint)
AUTHOR ADDRESS: Department of Microbiology-Immunology, Northwestern University Medical School, 303 East Chicago Avenue, Chicago, IL, 60611, USA**USA
JOURNAL: Journal of Clinical Investigation 109 (2): p233-241 January, 2002 2002
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LANGUAGE: English

ABSTRACT: Experimental autoimmune encephalomyelitis (EAE) is a Th1-mediated demyelinating disease of the CNS with similarities to multiple sclerosis. We and others have shown that a short-term course of anti-CD154 mAb treatment to block CD154-CD40 interactions can be used to prevent or even treat ongoing PLP139-151-induced relapsing EAE. However, little is known of the long-term effects of CD154 blockade on the development of antigen-specific T cell function. Here, we show that short-term treatment with anti-CD154 at the time of PLP139-151/CFA immunization inhibits clinical disease for up to 100 days after immunization. At this point, comparable numbers of Th1 cells are observed in anti-CD154 and control Ig-treated mice, as assessed by antigen-specific ELISPOT assays. Thus, the long-term Th1/Th2 balance is largely unaffected. Inflammatory responses are diminished in anti-CD154-treated mice, as indicated by reduced in vivo delayed-type hypersensitivity and reduced levels of splenic IFN-gamma secretion in vitro. However, upon adoptive transfer of T cells isolated from the spleens of anti-CD154-treated mice, these cells contributed as effectively to clinical disease as those obtained from control-treated mice. Thus, anti- ***CD154*** therapy leads to long-term therapeutic efficacy without exerting a long-term influence on Th1 development.

7/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)


14284021 BIOSIS NO.: 199800078268
Blockade of CD40-CD40 ligand pathway induces tolerance in murine contact hypersensitivity
AUTHOR: Tang Aimin; Judge Thomas A; Turka Laurence A (Reprint)
AUTHOR ADDRESS: Univ. Pa., 901-B Stellar-Chance Build., 422 Curie Blvd., Philadelphia, PA 19104-6100, USA**USA
JOURNAL: European Journal of Immunology 27 (12): p3143-3150 Dec., 1997
1997
MEDIUM: print
ISSN: 0014-2980
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ABSTRACT: Interactions between CD40 on antigen-presenting cells and its ligand (CD40L) on T cells has been implicated in T cell-mediated immune responses. Previously, we have shown that contact hypersensitivity (CHS), a cell-mediated cutaneous immune response in reaction to haptens, could be subclassified based on whether the hapten primed for Th1 or Th2 cytokines in cells isolated from draining lymph nodes. We also found that tolerance to a Th2-priming hapten could be induced only by simultane blockade of the CD40-CD40L and B7-CD28 at the time of sensitization. Here we demonstrate that blockade of ***CD40*** - CD40L signaling alone induces long-lasting unresponsiveness to the Th1 hapten 2,4-dinitrofluorobenzene (DNFB), and inhibits antigen- ***specific*** ***T*** ***cell*** proliferation in vitro. We find that CD40-CD40L signaling is required in the sensitization but not elicitation phase of DNFB-induced CHS, as treatment of mice with anti-CD40L monoclonal antibody (mAb) does not affect the response to hapten challenge in previously sensitized and untreated animals. Examination of cytokine production shows that anti-CD40L mAb decreases interferon-gamma production by draining lymph node cells from

DNFB-sensitized mice, and reciprocally increases interleukin (IL)-4 production. Consistent with this Th1 to Th2 immune deviation, anti-CD40L mAb prevents the induction of IL-12 mRNA in regional lymph nodes, an event which is normally seen within 12 h following hapten sensitization. In contrast, suppression of CHS by CTLA4Ig decreased the production of all cytokines by draining lymph node cells. Together, these data show that blockade of the CD40-CD40L pathway by itself is sufficient to induce tolerance to DNFB-induced CHS, and that this is associated with ***blockade*** of IL-12 induction and Th1 to Th2 immune deviation.

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